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The Management of Cranio-cerebral Injuries.*

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When an individual receives an injury to the head it is well to be cautious regarding the prognosis. Even though the patient appears normal, haemorrhage from the middle meningeal artery may be occuring and the patient will become dull in a few hours, and unconscious shortly afterwards. Or a patient with a severe laceration of the brain may seem well for forty-eight hours and then die in the next twelve. Several months after an injury all the symptoms of increased intra-cranial pressure may appear as the result of a venous haemorrhage on the surface of the brain, or later still the liquified and encysted blood clot may cause epilepsy, mental deterioration, headache, etc. Most often, of course, the individual who appears normal shortly after an injury remains well. Head injuries are now becoming so common that it behooves all of us to have a fairly satisfactory working knowledge of these cases.

The patient is often badly shocked when first seen. He will be white, cold, have a low blood-pressure, a fast pulse, and the breathing will be shallow and rapid. The first consideration must be the teatment of this shock, and this treatment may be carried out in the following way:

The patient is put between blankets with plenty of hot-water bottles; the foot of the bed is raised, hot coffee is given by rectum. The extremities are bandaged to support the peripheral circulation. Pituitrin and caffein may be given, but not morphia. Patients with cerebal injuries stand morphia very badly, especially if there is any tendency to respiratory failure. Futhermore, the morphia may mask subsequent important signs.

A chart of the respiration, pulse and blood-pressure should be commenced immediately, and charted every half-hour. If feasible a special nurse is obtained, also at this time certain general observations may be made which will not disturb the patient.

(a) Note if there is any obvious injury to the scalp or bleeding from the ears or nose.
(b) The patient's response to questions.
(c) Any obvious loss of movement in the limbs.
(d) The comparative size of the pupils.

After the Shock Has Passed off:

One should deal immediately with a laceration of the scalp and a possible underlying comminuted fracture.. The area is widely shaved. Anaesthesia is obtained by surrounding the area with an injection of 1% novocaine between the scalp and bone. If the edges of the laceration are crushed and soiled they are cleanly incised. Bleeding is controlled by turning back the aponeurosis of the scalp with a row of fine haemostats. No attempt is made to

*A lecture delivered to the fifth year students at the University of Toronto.

catch individual bleeding points. The underlying bone is carefully palpated and examined for exidence of fracture. If the fracture is a simple fissure, the wound in the scalp should be carefully sntured, by placing fine silk interrupted sutures sufficiently close to control haemorrhage. No drainage is necessary. If there is a depressed comminuted fracture, loose fragments of bone must be removed, as they are very likely to become infected. There is a certain urgency in dealing with these cases, as any infection may bring about a meningitis or brain abscess. If the dura is intact, under no consideration incise it in the presence of this infected field (fear of meningitis); if the dura has been torn, and the area is soiled, remove by suction and gentle swabbing any debris and obviously severely traumatized brain tissue. Only a small opening should be left through the dura, otherwise a large herniation of the brain may result. A cigarette drain of rubber tissue is left down to this area, and this should be removed in forty-eight hours, if there is no obvious evidence of infection. Any bone defect can be repaired at a later date if necessary.

The neurological examination is completed as soon as possible. If the patient has regained consciousness and is co-operating well, all the cranial nerves should be examined. The arms and legs should be tested for strength, changes of sensation, and reflexes. This record may be most valuable in determining treatment in the subsequent few hours or days. Many of these patients prove later to be medico-legal cases and one may be chagrined in court if one's record is not complete. If the patient remains unconscious, this record is necessarily curtailed. The chart of the respirations, pulse, and blood-pressure is carried on. A lumbar puncture is done and the pressure estimated. This pressure can only be recorded satisfactorily with a manometer; the rate at which the fluid flows through the needle is very fallacious. The normal pressure with a mercury manometer is from 7 to 12 mm. With the Ayer manometer, the pressure is recorded by the height of the column of cerebo-spinal fluid and is from 10 to 15 cms. If there is blood in the C.S.F., several ccs. are collected in three test tubes and if there is as much blood in the third test tube as in the first, one may assume that the bleeding was not caused by the puncture.

Stereoscopic plates of the skull, taken from both sides and from the front and back, are made when the patient can be safely moved. If the problem of diagnosis of compression from a torn middle meningeal has to be met, it is a great help if it is known whether a linear fracture is present which might have torn the vessel.

There may be bleeding or a cerebro-spinal fluid leak from the auditory canal (fracture of the base, through the petrous bone). If so, the external canal should be thoroughly cleansed with one in a thousand bichloride solution and a pledget of cotton soaked in this solution placed lightly in the canal, and changed every hour. Every attempt must be made to keep this area clean, as infection is very likely to set up a meningitis. The ear should not be syringed.

Having accumulated as much information as possible from the chart recording the respirations, pulse, blood-pressure, and cerebro-spinal fluid pressure, and having made as complete a neurological examination as possible, one is now in a position to consider whether the patient is suffering from concussion only, or whether he is showing signs of compression, or failure of the medullary centres.

Concussion:

This term may be very confusing, and it is not always clear just what is meant. It may be considered this way: if an individual receives a blow, he may be only dazed, and if one could see the brain, there would probably be no apparent microscopic changes. A more severe injury might bring about loss of consciousness, and examination of

the brain might show small microscopical haemorrhages scattered through it, and even gross lacerations. By concussion then we mean injury to the brain, which may only daze an individual, or render him unconscious. The injury might not be apparent even with a microscope, on the contrary there might be a gross laceration.

With this conception of concussion, the clincial symptoms vary according to the degree, and situation of the injury to the brain. Usually there is a period of unconsciousness which may be of short duration, or extend over many hours. Following this there is the stage of reaction frequently ushered in by vomiting and attended by various symptoms described at some length in the text books. The variation in the symptoms may be explained by the degree of interference with the physiological functions of the different parts of the brain; for instance, prolonged irritability from injury to the frontal lobes, convulsions from cortical irritation, paralysis or sensory changes from injury in the Rolandic area, vertigo from injury to the internal ear or cerebellar path-ways. Vomiting and abnormalities in the pulse, respirations or temperature from injury to the medulla.

Treatment of Concussion.

In addition to general symptomatic treatment and the avoidance of morphia, there should be absolute quiet and prolonged rest in bed. Luminol in two grain doses is a satisfactory sedative. As much as ten or twelve grains may be administered in twenty-four hours, usually by mouth, but it may be given hypodermically.

Compression.

By compression we mean that the brain is being compressed either by oedema or haemorrhage. There are certain signs which enable us to determine when this compression is taking place, and it is most important to be able to appreciate this and to institute proper treatment.

Compression due to extra-dural haemorrhage is almost always brought about by a tear of the middle meningeal artery. The usual syndrome of such a pathological condition is as follows. It is quite possible that the patient has had a fairly lucid interval after his initial injury, he will then commence to show the first and most important sign of compression, namely dullness and stupour which may go on to complete loss of consciousness. Loss of consciousness from concussion may be so prolonged that there is no lucid interval, and one then has to depend upon the other signs of compression:

1. The breathing becomes loud and stertorous and may drop down to four or five a minute. The patient can be heard breathing in the next room. Expiration and inspiration are about equal.

2. The blood-pressure rises.

3. The pulse becomes slowed and fuller.

4. The cerebro-spinal fluid pressure mounts.

(Hence the importance of keeping a chart of these readings right from the start, so that one may know beyond a doubt, that these changes are taking place).

Having determined that compression of the brain is taking place, one makes sure that a middle meningeal vessel is not bleeding and this involves a split vertical temporal muscle incision about a quarter of an inch in front of the ear, two inches long with the lower end at the zygomatic bone. If there is no bleeding beneath the temporal muscle, it is not likely that there has been a linear fracture which would bring about a tear of the vessel; however, it is not well to depend entirely upon this sign as it takes only another few minutes to take out a button of bone to see if there is an underlying clot. If there is a clot, the dura is not opened. The bone need not be replaced, the temporal muscle and scalp are quickly closed and the same procedure is carried out on the other side. A local anaesthesia with 1% novocaine

may be used, or if the patient is deeply unconscious no anaesthetic is required. If an extra-dural clot is found, the temporal muscle is retracted and the bone opening enlarged to two inches in diameter with rongeurs. The clot is then removed by suction and swabbing. With a good light, one is usually able to see the bleeding point, and can carefully pass a ligature around the vessel in the dura, taking care not to wound an underlying vessel in the brain. If a Cushing silver clip is available, it is much easier to use this in place of the ligature. However, if the bleeding point cannot be clearly seen, light packing with ganze will control the bleeding sufficiently, until one can get better facilities with which to work; even if the bleeding point is not stopped, the compression is relieved with the large opening in the bone. These patients die from compression, not loss of blood.

There are certain lateralizing signs which are of value in operating on a suspected middle meningeal haemorrhage:

1. The pupil on the side of the lesion may be large, due to paralysis of the third nerve, brought about by more or less direct pressure of the clot.
2. A linear fracture seen in the X-ray is a guide to the side.
3. There may be some weakness of the arm and leg on the opposite side.

It is important to operate on the other side, if nothing is found on the side first explored. Furthermore, remember that concussion with unconsciousness may be prolonged so that there is no lucid interval. In such a situation an exploratory operation is justified when there is evidence of compression.

If the compression is not due to extra-dural haemorrhage, it is likely brought about by a general oedema of the brain resulting from traumatism, and this may be treated in the following manner:

1. The return flow of blood from the brain is greatly facilitated by the respiratory movements of the chest. How disastrous to one of these unconscious patients to have him lying on his back with the respirations largely obstructed by the tongue. There is no more efficient method of increasing oedema of the brain and hastening death. The patient should be placed on his side and the tongue kept well forward, the head is elevated. It may be necessary to place tubes in the nares and inside the cheek to obtain a free airway.

2. Sufficient cerebro-spinal fluid is slowly withdrawn by lumbar puncture every two hours to reduce the pressure to half, that is, if the pressure is found to be thirty cms., it should be reduced to fifteen. The amount removed in thes punctures must be determined by manometer readings as it is possible to remove too much fluid and so bring about herniation of the medulla and cerebellum into the foramen magnum.

3. Two ounces of concentrated sulphate solution may be left in the rectum every two hours, or, if not retained, it is usually easy to introduce it into the stomach with a nasal catheter.

4. Concentrated solutions may be used intra-venously, probably the best is 75 cc. of 50% glucose. This hypertonic solution in the blood-stream brings about a flow of fluid from the brain to the blood-stream and so reduces the oedema. Glucose is better than sodium chloride which in the past has been commonly used in a 15% solution, sodium chloride solution quickly brings about a reduction in the oedema of the brain, but later the salt gets into the tissues and brings about a flow the other way. The glucose on the other hand (taken up by the liver and muscles), does not find its way into the brain and bring about this reverse flow. The concentrated glucose solution is conveniently obtained in 20 cc. ampules.

By these two forms of treatment, one surgical and the other non-surgical,

many patients suffering from compression may be saved by preventing failure of the medullary centres.

Failure of the Medullary Centres.

The medullary centres have been responding but they may commence to fail, and the picture is now changed. The pulse becomes rapid and irregular, the blood-pressure drops, the breathing although still loud becomes very rapid. This is the beginning of the end, and one can say almost with certainty that the patient will die.

The Simple Depressed Fracture.

There is no urgency about the treatment of this condition, as there is not the fear of infection that there is with a compound fracture. The fracture, however, should be re-elevated within three or four days. The procedure is usually carried out quite simply by turning down a flap of scalp and re-elevating the fragments piecemeal. Even if the fragments are detached from all blood supply they are left (note difference in compound fractures). It has been found from clinical experience that if these simple depressed fractures are not elevated the patient is likely to suffere subsequently from headache, epilepsy, or even have signs of pressure.

Summary.

In the management of cranio-cerebral injuries the important points are:

1. To treat the shock.

2. To record carefully from the start the necessary observations.

3. A laceration of the scalp is carefully dealt with.

4. A compound depressed fracture should be immediately and specifically dealt with after recovery from the shock.

5. Observation is maintained for the signs and symptoms of compression. Compression is usually due to (a) extra-dural haemorrhage (to be treated surgically), or oedema of the brain (see treatment).

6. Simple depressed fractures demand operation, but operation is not so immediately urgent as in the case of compound fractures.

7. Be guarded about the prognosis for at least three days even after seemingly trivial cases of concussion.

NOTE 1:—There is considerable controversy regarding the advisability of surgical intervention in cases of injuries to the brain giving definite localizing signs, and thought to be due to subdural haemorrhage, laceration or intra-cerebral haemorrhage. Only very exceptionally is it wise to operate on these cases. Sub-temporal decompression has been advocated in cases of compression thought to be due to oedema, and which cannot be controlled by the measures outlined. The turning down of large parieto-occipital bone flaps with the opening of the underlying dura and the replacing of the flaps loosely in position, has also been considered. Cerebellar decompression has also been suggested. It is certainly unwise in almost all instances to attempt any intradural surgical procedure when dealing with these cases.

NOTE 2:—The clean-cut, classical syndromes of concussion, compression and medullary failure are more than often not seen. One syndrome may follow the other so quickly that the picture is blurred. If the signs and symptoms of medullary failure follow quickly on those of shock and compression, very little can be done for the patient.

The Diagnosis and Treatment of Purpura and Allied Conditions.

By A. APPEL, 2T9.

(Continued from April number).

Purpura is described in the dictionary as a disease, characterized by the formation of purple patches on the skin and mucuous membranes, due to subcutaneous extravasations of blood. Strictly speaking purpura is not a disease but a symptom. Many patients are seen in hospital clinics and wards who show purpura either as their chief complaint or as an accompaniment of some other condition and it is somewhat difficult to say what the underlying cause is. Some of these patients are acutely ill and suffer from considerable loss of blood. In these cases it is essential that an early diagnosis be made and proper treatment instituted. It is with this idea in view that the various causes of purpura are presented and their treatment discussed, in this paper.

Diagnosis.

In the establishment of a diagnosis of purpura a careful examination of the blood is all important. This should include the following:—

1. Platelet Count.
2. Bleeding Time.
3. Coagulation Time.
4. Retractability of the Clot.
5. Leucocyte Count.
6. Examination of the Stained Smear.

Platelet Count.

This is by no means an easy matter since the greatest accuracy we can get is a count between 200,000-400,000. The platelets will adhere to the glass slide unless it is moistened, and to the test tube wall unless it is paraffined. We can learn much from a careful examination of the platelets in a blood smear. The smear must be perfect in all respects. All of the drop of blood must be used up, for if the drop is too large many of the platelets are carried off the end of the slide. If one is slow in starting the smear there will be a concentration of platelets at the beginning. A properly made smear shows most of the platelets at the end of the smear. They are not very common in the center of the smear and when found here they occur in one's, two's and three's, and not in large groups. They may occur in streaks. From the examination of a smear in such a manner one can say whether the platelets are normal in number, increased or decreased.

Another method which has given fair results, is to make a red blood cell count in the usual way, at the same time determining the relative number of platelets as compared to the red cells in the fresh specimen of blood. The number of platelets is then calculated.

The method of Wright and Kinnicutt is a direct method of counting the platelets and has given good results. Two solutions, one containing Brilliant Cresyl Blue and the other Potassium Cyanide, are used as a diluting fluid, and the counting is done in the ordinary counting chamber, but unless one has had experience with this method the results will not be very encouraging.

The platelet count is low in Essential and Symptomatic Thrombopenic Purpura but is variable in the other conditions giving hemorrhages into the skin.

Bleeding Time.

Is normal in non-thrombopenic purpura and in scurvy, and always prolonged in thrombopenic purpura from 20 minutes to several hours. The greater the reduction in platelets the longer will be the bleeding time. The bleeding time should be tested from sveral areas on the skin since the time required for the bleeding to stop may be normal in the ear and prolonged in the finger. The method commonly employed is to make

a needle prick in the skin and note the time required for the bleeding to cease, a piece of filter paper being touched to the drop of blood every 30 seconds. The bleeding time is always delayed when the coagulation time is prolonged (Morawitz). Prolonged bleeding time is seen in hemophilia, cholemia, and in Phosphorus poisoning, and E. T. Purpura.

Coagulation Time.

Is practically normal in the various forms of purpura. It is greatly delayed in hemophilia and coagulation may fail to occur in Fibrinopenia. The following methods are used:—

1. Collect 2-3 cc. of blood in a clean test tube and note the time that elapses before it becomes firmly coagulated so that the tube may be partly inverted without changing the position of the clot.

2. Another method is to collect about 5cc. of blood in an evaporating dish containing some lead shot and note the time required before the shot ceases to roll about.

3. A capillary glass tube filed about every one cm. is filled with blood from a needle prick. Then one segment of the tube is broken off at regular intervals and it is noted how long it requires for coagulation to occur. Coagulation has occurred when the blood can be drawn out into a thread between the two segments of the glass tube.

4. About 5cc. of blood are stirred with a fine glass red in an evaporating dish and the time is noted when the first thread of fibrin appears and when the blood is completely clotted. The normal is from 5 to 9 minutes.

Retraction of the Clot.

Normally when 5cc. of blood are collected in a clean test tube, we note that in 15 minutes after it has clotted the surface becomes concave. At the end of one hour it begins to be separated from the sides of the test tube. Retraction should be complete in 24-48 hours, when the clot is immersed in serum and remains attached to the sides of the test tube at only one point. At the bottom of the test tube is a scanty sediment of red blood cells. Retraction is due to the platelets. Irretractibility is a constant symptom of thrombopenic purpura. It is of great diagnostic importance as it occurs in no other type of purpura.

Leucocyte Count.

Should be carefully noted in all forms of purpura. It is of some value in differentiating Essential Thrombopenic Purpura from Symptomatic Thrombopenic Purpura. A low White Blood Count and low Platelet Count is in favour of the latter, while in Essential Thrombopenic Purpura, where the bone marrow is producing its constituents satisfactorily, the white blood count is normal.

Increased Permeability of the Blood.

In many patients with a purpuric tendency it is observed that a crop of petechiae arise on the arm after the application of the blood pressure cuff. This is called a positive Capillary Resistance Test and should be performed in the following manner:—

1. Apply the cuff of the blood pressure apparatus a hand's breadth above the elbow and produce a preessure midway between systolic and diastolic pressure, maintained for 5 minutes. Then examine the skin below the cuff after another 5 minutes for hemorrhages.

In scarlet fever and some other infectious diseases as well as in some cases of primary hypertonia and other conditions, a few tiny petechia form in the skin near the fold of the elbow. In Thrombopenic Purpura the hemorrhages are extensive. As a rule the lower the platelet count the greater the number and size of the hemorrhages. They may be so diffuse as to look like an erythema.

2. Trauma Phenomenon. Relatively mild blows with the reflex hammer over the sternum, radial border of the forearm, or on the tibis, will produce hemorrhages in the purpuric patients.

3. The injection of 1-2cc. of .2% sodium chloride subcutaneously will produce a hematoma in a susceptible person. The injury to the capillaries is the

same in normal and in purpuric patients, but in the latter the capillaries continue to bleed and produce a hematoma.

Characteristics of Purpuric Eruption.

They vary considerably in size and are larger in Thrombopenic Purpura than in other forms of Purpura. They occur in oval or round spots and ringed-shaped figures may be found, but are rare and suggest Anaphylactoid Purpura. The larger patches of purpura give the skin the appearance of a map. When they are less than 10 mm. in diameter they are called Petechiae, the larger ones Ecchymoses, and the extensive ecchymoses are called suggillations. The fresh eruption is bright red but soon becomes livid or purple. They cannot be obliterated by pressure which distinguishes them from mere congestion or from Naevi. Within a few days they begin to fade and change from bluish hues to brown-yellow, much the same as a black eye changes colours. In a week or two they disappear and leave only a yellowish stain. Successive crops are common.

Essential Thrombopenic Purpura.

Also called Purpura Hemorrhagica, of Werlhof's Disease, occurs in acute, chronic and intermittent forms. The average duration of an attack is from 1-2 months. The chronic variety is the commonest, and in this type they are subject to attacks for years, being well in between attacks. In this form hemorrhages occur from the nose and mouth, and into the skin. The blood changes are as follows:—

A. *Reduced Platelets.*

In this form of Purpura the count is usually below 100,000 and counts as low as 2,500 have been recorded. The severity of the hemorrhages is in proportion to the reduction of the platelets, and bleeding stops when the platelet count rises. In the intermittent form the platelets are normal in the interval, and in the chronic form they are continually low.

B. *Increased Bleeding Time.*

Here it is greatly increased but in the other forms of purpura it is normal. The bleeding time is usually looked upon as a measure of the functional activity of the platelets.

C. *Non-Retractibility of the Clot.*

The clot is firm but fails to contract and there is no extrusion of the serum as occurs in the normal.

D. *Morphological Changes.*

The blood picture is characteristic of a secondary anemia. The fall in hemoglobin is often much greater than the fall in the red blood cells. There is, in addition, an increase in the number of reticulated red blood cells (Minot). These are young red blood cells containing a granulo-filamentous substance stained by treating fresh blood with Brilliant Cresyl Blue.

Method—A drop of freshly-prepared Brilliant Cresyl Blue (1-4,000) is dried on a microscope slide; upon this dried stain is placed a drop of blood and a cover glass. 1,000-2,000 red blood cells are counted in a part of the preparation which is stained not too deeply or too lightly and the per cent of reticulated cells noted.

Normally the reticulated cells constitute about 1% of the cells. In Thrombopenic Purpura they number 10-19%. It is an expression of increased activity of the bone marrow. The platelets are often very large and may be distorted and degenerated. There is often a slight polymorphonuclear leucocytosis. This is of aid in distinguishing Essential Thrombopenic Purpura from Aplastic Anemia.

The diagnosis therefore depends on the following points:—

1. Bleeding from the mucuous membranes and into the skin.
2. Decreased platelet count.
3. Increased bleeding time.
4. Non-retractibility of the clot.
5. Normal clotting time.
6. Positive capillary resistance test.
7. There is usually an enlarged spleen.

II. Symptomatic Thrombopenic Purpura.

A. Blood Diseases.

Any of the so-called blood diseases may present purpura as a prominent symptom, and in some cases, particularly in lymphatic leukemias in children, extensive purpura may be the first symptom that anything is wrong. More often however the disease has already given rise to anemia or to enlargement of the spleen or lymph glands or to some other prominent symptom, and the diagnosis has already been made by the time the purpura supervenes. Acute Lymphoid Leukemia with leucopenia may be indistinguishable clinically and pathologically. Pernicious Anemia can be distinguished by the blood picture, as can also be Myeloid and Lymphatic Leukemias.

B. Infections and fevers.

Purpura may occur in any of the acute infections. It is more frequent in smallpox than in any of the other acute exanthemata. In the majority of the acute fevers purpura is more of prognostic rather than diagnostic importance. In diphtheria, for example, a single well-defined purpuric spot is a sign of grave omen but it does not assist at all in the diagnosis, which must be made on other grounds. The two fevers in which purpura is of value diagnostically are Typhus and Cerebro-spinal meningitis. The former is now very rare but when it was common and Typhoid fever began to be differentiated from it, the point upon which greatest stress was laid was that in true Typhus there is always Purpura, whereas in Typhoid all the spots fade on pressure. Purpura, occurring in the course of a meningitis, immediately excludes all forms of meningitis except the Cerebro-Spinal variety, although this form may occur without the presence of purpura. Lumbar puncture of course clinches the diagnosis. Smallpox may present cutaneous hemorrhages of three different types:—

a) There may be hemorrhages into the pustules in a late stage when the diagnosis has already been made and when the prognosis is not thereby made worse.

b) There may be hemorrhages between the pustules, vesicles, or papules, in which case the prognosis is not good.

c) There may be a hemorrhagic eruption all over the body in the prodromal stage of the disease in which case the patient will almost certainly die before the true smallpox eruption appears, so that if there is not an epidemic at the time the diagnosis may be difficult.

C. Intoxications.

Benzol and Arsenic may produce Purpura as a result of their toxic action on the blood and bone marrow, with the resultant decrease in platelets. Inquiry into the occupation or medications being taken should aid in establishing the diagnosis.

D. Anaphylaxis.

E. Avitaminosis.

Non-Thrombopenic Purpura.

A. Anaphylactoid Purpura (Schonlein-Henoch Disease).

In this condition there is an exanthematous eruption into the skin or mucous membranes, erythematous, urticarial, or edematous, as well as purpuric. On the mucous surfaces bleeding is usually accompanied by inflammation of the affected mucous membranes. Pains in and about the joints and abdominal colic are common. The blood platelets are little if at all diminished and may be even increased if the loss of blood has stimulated the bone marrow to activity. Bleeding Time and Retractibility of the clot are normal.

According to Pratt this is the commonest kind of purpura, there being 97 cases of this type in his series of 251 cases. Formerly the cases with joint pains were called Schonlein's Purpura or Purpura Rheumatica, and those with abdominal pains were called Henoch's Purpura. It has only recently been recognized that these are different types of the same disease. The exanthemetous

exudate and not the hemorrhage is the characteristic feature. In fact Purpura is often an unimportant symptom and may even be absent. A simple seizure may only last 1-2 days. Recurrence is the rule.

Symptoms—The onset varies. They may get headache, anorexia, and prostration. Arthritis often precedes the exanthematous rash by a few days and then the patient complains of pains in one or more joints. Occasionally abdominal colic ushers in an attack. The skin eruption is often unnoticed by the patient. There may be a temperature of 101-102.

Morphologically, inconstancy of the eruption is the rule. (Osler.) The eruption is an erythema and fades on pressure and true purpuric spots may or may not be present. The purpuric rash is often confined to the extensor surfaces of the legs, and the spots are smaller than in Essential Thrombopenic Purpura. Edema much like angio-neurotic edema occurs in some cases. In the joint type we find the knees and ankles most commonly involved. There is swelling, tenderness and pain, and the pain may shift from joint to joint. The arthritis is frequently the first manifestation and in a day or two the purpura appears, and the joint pains subside. Very often there are diffuse pains in the muscles of the arms and legs. In the abdominal type there are colicy pains, often agonizing, and form the most distressing feature of the attack. It does not yield readily to treatment and may not disappear for days. The pain is either around the umbilicus, epigastrium or hypogastrium. At the onset there is abdominal Rigidity and diffuse tenderness. The bowels are constipated and the condition may be confused with Acute Intestinal Obstruction. Vomiting occurs in a fair number and there is passage of a little blood in the stool, but never very much. The White Blood Count may be as high as 14,000. After a few days the gastro-intestinal symptoms cease, the purpuric spots fade, the arthritis disappears, and the convalescence is established. The attacks may recur. The average duration of illness is one month.

Acute Nephritis—Is the most serious complication. This is of the acute hemorrhagic type and occurred in 20 of a series of 43 cases in which abdominal symptoms were marked. (Pratt.) Of 7 deaths in a series reported by Osler, 5 were from uremia. The cases of nephritis fall into three groups:—

1. Acute course with dropsy and death in uremia in 33 months.
2. Albumin disappears and recovery occurs.
3. Those in which the nephritis becomes chronic.

Recovery is the rule although they may persist for 6 months or more. A mild hematuria may persist after the albuminuria has gone; albumin is usually abundant, and casts are absent or scanty. Bright's disease, especially of the chronic variety, may cause remorrhage anywhere in the body but is rare. Bamberger had 5 cases of Purpura in 2,340 cases of Renal disease.

Diagnosis—Is based on the following points:—

1. The occurrence of arthritis or abdominal colic with purpura, erythema, urticaria or edema. The purpura may be absent and the eruption is an exanthematous exudate.
2. Capillary Resistance Test is negative.
3. There is an increased susceptibility of the skin to chemical irritants such as tuberculin or caseosan.
4. The blood platelets are normal or only slightly reduced.
5. The retractibility of the clot is normal.
6. Bleeding and Coagulation Times are normal.
7. There may be a mild leucocytosis.

Because this form of Purpura may give severe abdominal colic, rigidity, etc., it should always be excluded before doing an exploratory laparotomy.

The prognosis in this type is fair, the only danger being from nephritis and

cerebral hemorrhage. Nearly all the others reocver.

B. Purpura Simplex.

Modern nosologists do not recognize the purpura simplex of older writers. The term was applied to those cases of essential or primary purpura in which there was no bleeding from the mucous membranes and little or no pain in the joints. In a few cases the platelets have been counted and found normal. (Duke, Pratt.) It is probable that many of the cases included under this name were mild cases of Anaphylactoid Purpura.

A purpuric eruption is usually the only symptom. The purpuric spots are as a rule small and the eruption symmetrical on the arms and legs. A few cases show spots on the face. The disease is generally afebrile and there may be slight pains in the muscles and joints. If the arthritic manifestations are at all marked the case should be classed as an Anaphylactoid Purpura. Slight nausea may usher in an attack, but diarrhea is rare. Albumin may occur in the urine. The duration is 1-6 weeks. The eruption comes out in crops.

C. Purpura Fulminans.

Is a rare condition. The ecchymoses spread with startling rapidity and within a few hours an entire extremity or the greater part of the trunk may assume a blue or reddish black colour. It is usually fatal in 18-48 hours and no patient has recovered. All the typical cases have been in children. There is supposed to be a marked thrombopenia although normal platelets were found in some. A diminution in neutrophilic leucocytes has been noted.

D. Avitaminosis.

In Scurvy there occurs a brawny hemorrhagic infiltration of the thigh or lower leg, the Scorbutic scleroderma. There are petechiae in the skin and characteristically around the hair follicles. Bleeding from the gums may occur in both Scurvy and Thrombopenic Purpura but in Scurvy the gums are spongy. There is usually some deficiency in the diet, and the platelet count is normal.

E. Capillary Weakness.

1. Senile Purpura—Is seen as purpura along the outside of the forearm in successive dark purple irregular blotches. It has also been called Purpura Cachectica. The backs of the hands and forearms are more frequently involved here than in any other form of purpura. It is relatively rare

2. Mechanical Purpura—Purpura appearing after an epileptic seizure or whooping cough, are of this type. Petechiae are sometimes produced in the knee by tight bandages on the thigh. They are due to lesions in the capillaries of the skin and are a form of vascular purpura. The fact that the eruption in almost every form of purpura is more abundant on the legs than elsewhere, is probably the result of the mechanical influence. Attempts to sit up after an attack of purpura may be followed by the appearance of a new crop of petechiae on the legs. Archard and Gronet have given this the name of Purpura Orthostatique, and Pratt describes such a case.

F. Infection—Bacterial capillary emboli or toxic capillary injuries in septic disease.

Almost any condition in which there are pyogenic organisms or their toxins circulating in the blood, may be associated with purpura, and this applies to pyemia and septicomia in general. The diagnosis is made by blood culture although a negative culture does not exclude them. There will very likely have been rigors, fever, and other symptoms pointing to the nature of the case.

1. Ulcerative Endocarditis—Seeing that it is very rare to get purpura in association with chronic valvular disease of the heart, if both Purpura Rheumatica and Subacute Bacterial Endocarditis can be excluded, the occurrence of purpura in a heart case may be one of the main symptoms indicating that an ulcerative endocarditis has supervened. The

purpura may be general or local, the former when there is hemolysis, and the latter when there has been a splutter of minute peripheral emboli. Other symptoms of the disease help to confirm the diagnosis.

2. Pyorrhea Alveolaris—Is another condition which leads occasionally to extensive purpura and other hemorrhages, and sometimes the patient developes Purpura of obscure origin, when the real cause lies in infected tonsils, especially when these are the persistent habitat of the streptococcus hemolyticus.

3. General Tuberculosis—Is not a common cause of Purpura, and yet in a few instances extensive purpura has been the first and for the time being the only symptom of an obscure illness which has ultimately turned out to be General Tuberculosis. The patient has generally been a child and the diagnosis has only been possible when the course of the case has been watched. The same may be said of certain cases of sarcomatosis.

G. Drugs and Poisons.

Many drugs produce Purpura but none do so commonly. Iodine heads the long list of these drugs. The iodides produce Purpura only in persons with idiosyncracies. Grossman describes a case which had Purpura and Epistaxis following iodides. S. Mackenzie reported a fatal case in an infant 5 months old after 2½ grains of Potassium Iodide. At the Boston City Hospital they had a case following the administration of 180 grains of Potasium Iodide over a period of 4 days. The patient's fingers were swollen and covered with hemorrhagic blebs and there were purpuric spots on the thighs. Osler also reports a case following iodides in which there was an Urticarial Rash, Purpura and Fever. Several cases have been reported due to Mercury, Antipyrine, Chloral, Copaiba, Quinine, Belladonna, Arsenic, Turpentine, Phenscotin, Ergot, Salicylic Acid, and Snake Venom.

F. Due to Local Injuries.

1. Flea Bites are by far the commonest cause of Purpura seen in the Out-Patient Department of a hospital, and they may sometimes be so numerous as to raise a misleading suspicion that the patient is suffering from some more serious disease. The relatively small hemorrhagic foci, and their prevalence on the parts covered by the clothes rather than on the hands, face or exposed parts of the legs, serve to indicate the diagnosis. Even in a severe case the examination of the purpuric spots under a magnifying glass reveals the central puncture of the bite.

2. Pediculosis—Most commonly produced by Pediculus Corporis or Vestimentorum. The circumstances of the case, the distribution of the Purpura itself, and the marks of scratching, particularly in the region of the collar or where other constrictions in the clothes occur, would indicate the diagnosis.

3. Bug Bites—Will be suggested by the circumstances of the case, and the central puncture. In susceptible individuals the purpuric areas may be a centimetre or more in diameter and are often purplish or bluish, rather than bright red.

4. Leech Bites—The hemorrhage around a leech bite is so characteristic that once seen it cannot be mistaken for anything else.

5. Blows and Sprains—If sufficiently severe, produce Purpura even in healthy individuals, in whom the history gives the diagnosis. Some normal individuals, particularly women, bruise with such ease that there may be no clear evidence of injury unless careful inquiry is made. Such easy bruising may also occur in any of the blood diseases.

5. Epilepsy—A case of epilepsy may sometimes come under observation for multiple bruises simulating some form of purpura, but due to injuries produced during attacks, which may themselves be unsuspected if they occur during the night.

6. Hysteria and Malingerers—Very extensive Purpura has been produced on the legs or other parts by multiple self-injury in girls suffering from Hysteria, or by Malingerers. Serious organic dis-

ease may be feared unless other factors are in the case or the distribution of the Purpura suggests an artificial origin.

7. Spontaneous Rupture of a Muscle Leads to extensive purpuric extravasations of blood, but the diagnosis is not difficult if the history is clear. One can often feel the place where the muscle has given way, unless it is too deep-seated, *e.g.* the Plantaris Longus, which is apt to rupture during the sudden efforts, such as may be made in playing tennis, etc.

8. Varicose Veins—The purpuric discoloration of the skin around varicose veins, together with its resultant dark brown pigmentation, is familiar to all.

G. Endocrine and Nervous.

There are a fe weases in the literature in which purpura is associated with the menstrual function. One woman vomited and had Purpura every three weeks, but did not menstruate. Cazenave observed a periodic monthly Purpura in a girl who never menstruated. Trousseau reported a case of Purpura Urticans eruption, accompanying each menstruation for a year or more. Purpura occurring during pregnancy and childbirth probably do not belong to this group.

There are well-authenticated cases of Purpura developing after severe fright in persons of nervous temperament. Weir Mitchell reported 3 cases characterized by severe neuralgia and muscular spasms, in which purpuric spots appeared at or near the painful points. Bouchard has observed Purpura in cases of Trigeminal Neuralgia and others in sciatica. Purpura has been described in connection with lightning pains of Tabes (Straus), in hemiplegia on the paralyzed side (Gilber), and in multiple sclerosis (Chevalier).

II. Jaundice.

This should be born in mind as a cause for Purpura, for, although the occurrence of the latter does not assist in differentiating one kind of jaundice from another, one might be misled into diagnosing something more serious than is necessary. One did not bear in mind that any kind of jaundice may produce Purpura. Moreover, some of these patients may appear to have been grossly ill-treated if one were to judge only by the degree of bruising that may result from ordinary palpation. The danger of fatal oozing after operation is always to be remembered when surgical measures are thought of in a jaundiced patient. Spontaneous hemorrhage into the skin is less mommon here than in hemorrhage from what would be otherwise trivial causes.

1. Chronic Alcoholism.

Purpura may develop in chronic alcoholism, especially if it has already led to cirrhosis of the liver or peripheral neuritis. It is generally confined to the legs below the knees. In many cases the diagnosis is easy even if the history is not given with perfect honesty.

IV. Hemophilia.

The essential characteristic of hemophilia is the occurrence of severe hemorrhages following such slight injuries that in a normal person little or no bleeding would result. There is no tendency for the hemorrhage to stop in the usual manner. Epistaxis is common. Spontaneous hemorrhages do not occur even though the patient may not remember the injury. The most trivial injury such as pressure of the hand on the skin, or the rubbing of the clothes on the body may produce hemorrhages. The skin lesions vary from a bruise to a hematoma. Ecchymoses and suggillations are common.

The tendency to bleed is always chronic and is noticed early in childhood. If adult life is reached the bleedings may decrease in frequency and severity. Variations in the liability may occur from week to week. Addis divides the cases into 3 groups:—

1. Those who are never free from hemorrhages into the skin or elsewhere.

2. Those in whom trivial accidents do not lead to hemorrhages.

3. Those in whom bleeding persists for longer than the normal, but the trauma is great enough to produce a hemorrhage in the normal person.

The severity of the case is dependent on the retardation of the Coagulation Time. The real distinction between a Hemophiliac and a normal, is not the occurrence but the amount of bleeding. In a normal person when the skin is traumatized, a few capillaries are injured, but are soon sealed up again. The Hemophiliac continues to bleed, tlthough the hemorrhage at the moment of injury is no greater.

The subcutaneous hematomas are sometimes painless, but may give rise to much discomfort, and gastro-intestinal disturbances. They may reach a very large size and weaken the patient from loss of blood, and they may even die from this subcutaneous hemorrhage. Hematomas tend to spread far from their point of origin. The pressure of extravasated blood on nerves may give severe pain or paralysis. The pulse may be obliterated because of arterial compression. Cyst formation may occur in place of the usual absorption. A hematoma may closely resemble an abscess, but when incised, blood instead of pus is obtained.

A trivial wound or a mere scratch may be followed by a more severe hemorrhage than a clean cut. Fatal hemorrhages have occurred during operations upon bleeders. The attempt to amputate the bleeding part, or tie an artery in case of uncontrollable hemorrhage has been followed by death. The blood drips from a wound as it would from a soaked sponge.

The hemorrhage usually lasts for hours and may continue for days or weeks. Litten mentions the case of a bleeder who in 11 days lost 24 pounds of blood. Bleeders have been observed to recover from almost lethal hemorrhages with extraordinary rapidity. After severe long-continued hemorrhages the blood may be almost colorless. Wounds in bleeders often heal very slowly and absorption of the hemorrhage is retarted.

Joint Affections—Are very common in Hemophilia. There occur all the different joint affections from the mildest arthralgia to the severest hemarthroses, and are due to bleeding from the synovial membrane into the joint cavity. It never occurs in some Hemophilic families. Three stages are described::—

1. Hemarthrosis, the stage of hemorrhage.
2. Inflammatory stage.
3. Contraction and scar formation, i.e., ankylosis.

A joint may become swollen in 5-10 minutes from hemorrhage. There may occur complete absorption and no loss of function. The knee and elbow are most frequently involved but other joints may suffer. Pain may or may not accompany the joint swelling. Fever is often present. Spontaneous dislocation of the joint may occur.

Diagnosis.

1. The condition is hereditary and manifests itself in childhood.
2. Coagulation Time is delayed.

In a suspected case the blood should be obtained from a vein with a needle, the capillary blood from a skin puncture not being good enough, because it may not show the true Coagulation Time. In one case reported by Howell the Coagulation Time of the blood from the skin was 10 minutes and from the vein 4½ hours. Very often, however, the blood from the skin puncture gives adequate results. In 12 cases studied by Addis, the C.T. was 15-85 minutes and in 9 of these it was over 54 minutes.

3. It usually occurs in males and is transmitted by females only.
4. Prolonged Bleeding Time.
5. Normal Retractibility of the Clot.

Hereditary Pseudo-Hemophilia.

14 cases of this condition are reported in the literature. Males and females are equally affected. The platelets are normal in number but not in appearance, there being apykrosis and a loss of granules in the platelets. Retraction of the clot is incomplete, and the Bleeding and Coagulation Times are normal.

Hereditary Thrombopenia.

Five cases have been reported. It may occur in males or females, and may be transmitted by the father. The platelet count is low, the Bleeding Time is increased, and the Coagulation Time is normal. The Capillary Resistance Test and the Puncture Test are positive.

Fibrinopenia.

Or Sporadic Pseudo-Hemophilia, is a condition in which there is a complete lack of fibrinogen in the blood, and they bleed from minor injuries. The platelets are normal and the blood does not coagulate spontaneously, even after the addition of serum tissue extract or platelets, because of the absence of fibrinogen. There have been two cases reported, one of which bled to death following a needle prick on the finger.

Bernuth's Pseudo-Hemophilia.

Greatly resembles hemophilia. It is diagnosed by means of the capillary microscope. The capillaries are seen to remain open and continue to bleed, instead of closing as they normally should. The platelet count and bleeding time are normal, but the coagulation time is prolonged.

Hemorrhagic Disease of the New-Born.

A tendency to bleed is seen in the first few days of life, in many conditions, especially in those of an infectious character like syphilis or pyemia. With most of these the hemorrhages are small and the condition may be compared to the hemorrhagic tendency seen in certain infectious diseases of later life, such as measles, smallpox, bacterial endocarditis, etc. There are a class of cases, however, in which hemorrhages are not associated with any other known process, and in which the escape of blood from the small blood vessels is the chief symptom. In these cases the bleeding is much more extensive than in the others mentioned. These hemorrhages are characterized by the fact that they are spontaneous in origin, having no connection with delivery, are multiple in location, and tend to cease spontaneously after a limited time, but are often greatly influenced by treatment. This is called the Idiopathic Hemorrhage of the New Born or Morbus Hemorrhagica Neonatorum. The skin is most frequently involved, the gastro-intestinal track next, and all authorities stress the occurrence of melena. In addition to signs of hemorrhage there are signs of anemia. Melena should not be confused with the expulsion of meconium. The latter shows no red blood cells microscopically, and leaves no red halo on the diaper. Hemorrhagic disease should not be confused with Hemophilia, which rarely becomes manifest till one year of age, whereas the former always occurs in the first few days of life. Hemophilia is an hereditary disease transmitted by the females of a family, manifesting itself in the male members of the family, and continuing all through the patient's life. Morbus Hemorrhagica Neonatorum occurs equally in both sexes, and is limited by days, does not recur, and is not, so far as we know, hereditary.

TREATMENT.

Essential Thrombopenic Purpura.

The patient should be kept in bed for some time after the petechiae cease to appear, and the attendants should handle the patient lightly and with care. The diet should be light and varied during the disease, and foods rich in iron should be given during convalescence. The following drugs have been recommended but are not of very much value:—

1. Aromatic sulphuric acid, min. 15-30 ex aq.
2. Oil of turpentine, min. 15 t.i.d.
3. Ergot.
4. Calcium chloride and Calcium Lactate.

Transfusion is of the greatest value if there has been much loss of blood. It may stop the bleeding completely. 200 cc. of defibrinated blood may also stop the bleeding which indicates that the pres-

Condition	Platelet Count	Bleeding Time	Coagulation Time	W.B.C.	Retract-ability of Clot	Capillary Resistence Test	Other Factors
Essential Thrombopenic Purpura	Decreased	Prolonged	Normal	Normal	No.	Pos.	No apparent cause. Enlarged Spleen.
Symptomatic Thrombopenic Purpura	Decreased	Prolonged	Normal	Decreased	No.	Pos.	Some cause such as a Blood Disease, Benzol, Infection, etc.
Anaphylactoid Purpura	Normal or Slightly Decreased	Normal	Normal	Slight Inc.	Normal	Neg.	Arthritis or Colic. Erythematous skin eruption. True Purpura may or may not be present. Sensitive to proteins.
Non Thrombopenic Purpura	Variable	Normal	Normal		Normal		Usually a definite cause like drugs, injuries, Vit. deficiency, etc.
Hemophilia	Variable	Prolonged	Greatly Prolonged		Normal		Is Heredity and Manifests itself in childhood. Occurs in Males and transmitted by Females. Joints often involved. Rarely starts before 1 yr. of age.
Fibrinopenia	Variable		Greatly Prolonged May not occur.				No Fibrinogen in Blood.
Hereditary Psuedo Hemophilia	Normal in number. But abnormal in appearance	Normal	Normal		Incomplete		Males and Females both affected.
Herditary Thrombopenia	Decreased	Prolonged	Normal			Pos.	Occurs in Males and Females. May be transmitted by Father.
Bernuth's Pseudo Hemophilia	Variable	Normal	Prolonged				Diag. by means of Cap. Microscope-capillaries are seen to remain open and continue to Bleed.
Morbus Maculosa Neonatorum							Occurs in 1st few days of Life. In M. & F. Is not hereditary. Lasts only few days. Does not recover.
Scurvy	Normal						Diet deficiency (Vit.). Gums Bleed. Gums are spongy.

ence of platelets is not necessary to check bleeding. . The serum should be kept in the icebox for 24 hours to avoid a reaction. Williamson has produced a rise in the blood platelets by the injection of 30 cc. of citrated blood every week. At Yale favourable results were obtained following the use of Mercury Vapour Quartz Lamp, after which the platelet count rose and the bleeding ceased. Hirsch was able to influence local bleeding, especially from the nose, by means of diathermy. There is a report by Sauer on the use of "Vivocoll," or bovine plasma, to which has been added sodium citrate solution and a preservative. Before use Calcium Chloride solution is added. This preparation seems to act as an excellent hemostatic agent on wound surfaces. Pectin, prepared from various flowers and fruits, has definite hemostatic powers. It has no action in vitro, but in most animals and in human beings it seems to reduce the coagulation time when given by mouth, but more rapidly when given subcutaneously.

The treatment par excellence which has come to light in the past few years, is that of splenectomy. In 1916, Kaznelson, a surgeon of Prague, observed that the spleen was enlarged in 2-3 of the cases of Purpura, and thought it might be evidence of excessive thrombolytic action of the spleen. He put the question to the test by removing the spleen from one of his patients, a woman who for 10 years had suffered from Purpura with epistaxis and menorrhagia so severe as to endanger her life on many occasions. Splenectomy resulted in a dramatic cure which 7 years later he reports is still maintained. Since then many have been operated upon successfully. Only the chronic cases should be done. The mortality in those is only 5%, while in the acute forms it is much higher. Coincident with the post-operative rise in platelets, the hemorrhages cease, the bleeding time becomes normal, and the clot retracts. In many cases the ultimate level of the platelet count is subnormal, but it remains high enough to prevent recurrence of bleeding.

Anaphylactoid Purpura.

Treatment is unsatisfactory and is largely placed on hypothetical grounds. They usually recover speedily from an attack but recurrences are common and cannot be prevented. Animal charcoal, 20-30 grains, has been given, with the object of combating a hypothetical poison of the gastro-intestinal tract. Rantenberg thinks it is a tropho-neurotic disturbance in the lower cord and cauds equine, and induces lumbar anesthesis with excellent results. He injects 4% solution of novocasin in sodium chloride solution. Fair results have been reported from the careful injection of foreign protein into the skin. Frank begins with 1/10-1/1,000 part of the smallest dose of foreign protein material that produces a local reaction, and slowly increases it. He uses Old Tuberculin, Yetren-Casein, or milk. Calcium to restore the capillary wall to its normal state has been used. 30-75 grs. by mouth may overcome the intestinal colic. To get a general reaction on the capillaries of the body give 25 cc. of Calcium Chloride intravenously once or twice a day.

Hemophilia.

Every hemorrhage in hemophiliacs endangers their lives but bleeders rarely die from their first hemorhage. Childhood is the most dangerous period. With sufficient care we can get these children to adult life and the older they are the greater is the tendency to outgrow it. Past experiences and the family history will help in forming a prognosis.

1. Preventive Measures—An operation should never be performed upon a patient until it is ascertained that he is not a bleeder. In hemophilic families the children should be protected from injuries from birth on. Too active games like running, jumping, etc., should be forbidden. The teeth should be attended to proceded by a prophylactic transfusion if necessary. The disease is rare in warm climates, therefore a trip to the south should be advised during the winter if it is possible.

2. Local Measures. Plugging the nostril and packing the uterus may help. Ferric chloride or gauze soaked in adrenalin are not much good. The only tissue extract of any value is Cephalin. A gauze saturated in Cephalin and put up in vacuum-sealed tubes for application to bleeding surfaces has been prepared. It deteriorates when exposed to air, and it is this reaction which plays a part in stopping the bleeding. The local application of human blood has proved fairly satisfactory, serum not as much so. Animal tissue extracts have proved beneficial. The extract is made by grinding up liver in NaCl sol. The extract must be cloudy. Gauze soaked in it is applied to the bleeding surface with good results.

For fresh bleeding into a joint elevate the part and supply cold compresses. Later apply a compression bandage and after 3 or four days start gentle massage and movements. If ankylosis is inevitable get the limb in good position.

3. General Measures. 10-20 cc. of fresh serum intravenously or 20-30 cc. of into the tissues is of value. Human serum is preferred, but animal serum works. It results in a temporary shortening of the coagulation time and must be repeated in 2 or 3 days. This method is not as popular as it used to be. Witte's Peptone is hegarded by some as more efficient. A solution is made up as follows:

Witte's Peptone, 5 gms.
Sodium Chloride, 5 gms.
Distilled Water, 100 cc.

Sterilize and inject 10-20 cc. subcutaneously. It may cause pain at the site of injection and some fever. Report every 2 or 3 days for a week or two if necessary. Before beginning a second series of injections an interval of 5-6 weeks should elapse. Radiation of the spleen has been recommended by Stephan. Euphyllin, according to Nonnonbruch, shortens the coagulation time. The injection of sodium citrate, the ingestion by mouth of considerable quantities of fibrinogen, and the irradiation of different parts of the body have been recommended by various workers. Transfusion is by far the best method yet. Citrated or uncitrated blood may be used. The C.T. is reduced for only a few days. Minot and Lee brought the C.T. down from 1½ hours to 8 minutes in one case, but it completely returned to its original level in 92 hours. Minor operations and extraction of teeth could be done during this time. The patient should be transfused again in 48 hours and even a third time if necessary.

Hemorrhagic Disease of the New Born.

The treatment is blood transfusion 15 cc. per pound body weight being used, and the donor should be of the same group as the child. This treatment not only supplies the lacking constituents of the blood, but replaces that lost by the hemorrhage.

A Brief Synopsis of the Treatment of Lung Abscess.

By JOHN HEPBURN, M.B.
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The usual precursor of lung abscess is an operation on the nose or throat, but many cases follow pneumonia and a few are due to infected emboli lodging in the pulmonary circulation.

Chronic bronchitis occasionally results in lung abscess and at times lung abscess appears to develop spontaneously.

Clinically lung abscesses may be acute or chronic and as the results of treatment are often excellent in the former but very disappointing in the

latter, it is most important to make an early diagnosis and institute treatment promptly.

In a typical case following tonsillectomy for example, a few days after the operation the patient develops the signs of pneumonia and a day or two later, the patient or physician may notice that the patient's breath has become very foul and that the odour of the sputum is also offensive.

Clubbing of the fingers and toes may appear in a week or two and is occasionally preceded by uncomfortable tingling in the finger tips. The sputum becomes more offensive and the need for expectoration often disturbs the patient's sleep.

The patient often discovers that certain positions of the body increase the coughing and expectoration and the other positions allow him to rest quietly. This depends on the relation of the abscess to the bronchus which drains it.

The physical signs depend on the size of the abscess and the relative content of air and pus, but on the whole the signs are those of consolidation and cavitation, for the abscess always has an area of inflammation around it.

Radiographically there is seen an area of increased density surrounding an opaque collection of fluid with an upper level which is horizontal in all positions of the body providing air is also present in the abscess cavity.

An acute lung abscess quickly becomes chronic and pleural adhesions form, limiting movement of the lung, and as the sufferer from chronic lung abscess is a source of perpetual disgust both subjectively and objectively on account of the extremely offensive odour, the physican's problem resolves itself into preventing the condition from becoming chronic.

Treatment of Acute Lung Abscess.

The diagnosis having been made two lines of treatment, or a combination of both methods may be tried.

The methods which give the best results are: postural drainage and artificial pneumothorax.

Postural Drainage:

This consists in finding in which position of the patient's body the abscess empties by mouth most freely. The position for the given patient is unpredictable as it depends on the relation of the cavity to the bronchus leading into it. Usually the patient finds that leaning, head down, over the edge of the bed with the weight taken on the arms resting on a low chair, and the body tilted to right or left is most efficacious.

Having discovered the best position for drainage, the patient should be made to assume that position several times a day—the number of times varying with the amount of discharge. Each period of drainage should last about half an hour, and the last period should be just before settling down for the night, so as to give the patient the best possible chance for unbroken sleep.

A carefully charted record of the daily amounts of sputum should be kept as an index of the success of the treatment. Progressive diminution of the daily discharge, coincident with clinical improvement, is what one hopes for and means that the cavity is becoming smaller.

Artificial Pneumothorax.

Collapse of the lung (in which the abscess lies) by the introduction of air into the pleural cavity, is the method which has given the best results in cases of acute lung abscess at the Toronto General Hospital, providing the treatment has been started early.

The absence of pleural adhesions, or the presence of few or recent adhesions and the more or less central position of the abscess are essential for successful treatment by this method. If the adhesions are multiple, strong or widespread, satisfactory collapse of the lung abscess cannot be obtained as a rule. However, fairly strong adhesions may gradually stretch and finally give way if by repeated introductions

of air into the pleural cavity, the pressure there is progressively raised.

If the abscess is in the base of the lung close to the vertebral border, and if the lung there is bound down to the diaphragm or chest wall, collapse of the rest of the lung may lead to the drainage from the abscess being shut off, and the patient will be worse off than before.

The most satisfactory results in cases of acute lung abscess at the Toronto General Hospital, have been obtained by early repeated pneumothoraces combined with postural drainage.

On the other hand, if the abscess is close to the pleural surface of the lung, stretching of adhesions may result in rupture of the abscess into the pleural cavity. This of course means an empyema, a most distressing and difficult complication to deal with.

Chronic Lung Abscess.

As pointed out before, dense pleural adhesions soon form, limiting the movement of the lung and making the satisfactory collapse of the lung by pneumothorax an impossibility. Medical treatment holds little hope of improvement and consists in postural drainage and an attempt to improve the general condition of the patient by good feeding and exposure to sunlight. The offensive taste and odour often make it difficult for the patient to enjoy meals.

Bronchoscopy.

Certain hospitals report good results by aspiration of the cavity together with the introduction of antiseptics through a bronchoscope in the hands of a good bronchoscopist.

Surgical Treatment:

Like medical treatment the results of surgical intervention are better the earlier the operation is done. Probably the best time for surgical measures is immediately after medical treatment has failed to result in a cure and before the case becomes chronic; when the abscess is surrounded by dense inelastic fibrous tissue which cannot be collapsed.

The best surgical results are in early cases and the method of choice at present is drainage of the abscess after a wide resection of the chest wall — the path for drainage being made to the abscess cavity by an electrocoagulation needle.

The desideratum is a cone-shaped cavity with the apex at the depth of the abscess and the base formed by the opening in the chest wall. This is to make the cavity close from within outwards.

In addition to surgical drainage a more or less extensive thoracoplasty may be done to diminish the size of the lung and abscess cavity. In the hands of a skilled and experienced surgeon the results of this method of treatment are quite good in early cases and the operation mortality is not high but long standing bronchial fistulae at times result.

Lobectomy i.e. removal of the lobe of the lung containing the abscess is attended by too high an operative mortality at present to be much in vogue.

Summary:

Lung abscess is a sequel of operations on the upper respiratory tract and of pneumonia, etc.

The early symptoms and signs are those of pneumonia. Then suddenly the malodoriferous evidence of putrefaction appears in the shape of most offensive sputum and breath.

The all-important factors in the successful treatment of lung abscess are first, the early diagnosis of the condition and secondly the immediate institution of vigorous treatment in the shape of artificial pneumothorax combined with postural drainage.

The failure of the physician to diagnose and treat such a case early is calamitous as the results of treatment when the condition becomes chronic are much less satisfactory and the patient often becomes an object of horror to all in attendance until death by pneumonia or inanition brings release.

The Quicksilver Doctor and Robinson Crusoe.

By Miriam A. Brick, 3T0.

Thomas Dover, doctor, has drifted into immortality and the life of the modern medical student on the potent yet palatable powder by the same name; while Captain Dover erstwhile buccaneer and discoverer of the original Robinson Crusoe has been obscured by the passing years.

Regarding the facts of Dr. Dover's life little is known. History relates that he came of an interesting and original family and that his grandfather, Robert Dover, was the originator, at the beginning of the 17th century, of the Olympian games, held in the Coltwold Hills as a protest against the increasing puritanical prejudices. Born in Warwickshire, 1660, we find little or no account of him till he appeared at Cains College, Cambridge, from where he obtained a medical degree in 1683.

After taking his degree Dover settled in Bristol. Here during an epidemic of spotted fever—1696—he emerges from obscurity as the first medical man who gratuitously offered his services on behalf of the poor under the care of the city. The practise in Bristol was short-lived. After accumulating a small sum of money Dr. Dover associated with Alderman Bachelor and Sir John Hawkins as promoter in a privateering venture. During this period buccaneering was accepted as a legitimate occupation since it appealed to the sporting instincts by promise of adventure and excitement with high stakes.

Of this memorable voyage we learn it was one of the best organized and equipped expeditions of this sort that ever left England. Two boats, the Duke and Duchess, were fitted. Chief in command was Captain Woodes Rogers with one of the most famous old south sea captains, William Dampics, as pilot. Captain Dover was third in command and because of his large financial interest was allowed a double voice in all councils.

Captain Rogers in "A Cruising Voyage Around the World" provides a complete description of the finding of "Robinson Crusoe." The Duke and Duchess reached Juan Fernandez, February 1st, 1710. That evening the crew observed a light on shore and were apprehensive of French ships or perhaps a Spanish garrison. However, on the following day since no ships were visible, Captain Dover accompanied by six men set out for shore in the yawl. When they had been absent for several hours alarm was beginning to spread among the men, but soon the yawl returned carrying an abundance of cray-fish and an individual clothed in goat skins who appeared wilder than the original wearers of the skins. The man told the members of the expedition that he was Alexander Selkirk, a Scotsman who four years previously while sailing with Captain Straddling had been left behind on the island as the result of an argument with the captain in regard to the leaky condition of his boat.

After the first few months during which the loneliness was extremely depressing Selkirk had found life on the island interesting and stimulating. His complete outfit consisted of his clothes, bedding, firelock, some powder, bullets, knife, kettle, Bible, some books, a little tobacco and his mathematical instruments. Two huts were soon built of palm trees, thatched with long grass and lined with goat-skins. When no powder remained the only means of making fire was to rub two sticks together. During the four years he had killed 500 goats while many others were caught, marked and allowed to go. When all the ammunition had been used Selkirk developed remarkable

speed, and running after the goats would overtake them by mere fleetness of foot. On the departure of the ship it was agreed that Selkrik would serve as mate aboard the Duke.

Subsequently the expedition sacked Guacaquil in Peru, while in the city the sailors stored their plunder and slept in the churches where they were tremendously disturbed by the smell of recently burned corpses of the victims of a plague epidemic. Twenty-four hours after their return to the ships 180 men developed plague. Dover ordered bleeding and the four surgeons went to work taking about 20 ounces of blood from each man. Following this dilute sulphuric was given the patients to drink. Fortunately the cases were fairly mild and they escaped with only eight deaths.

The expedition reached England in 1711 carrying plunder to the value of £170,000, of which Selkirk received his share. Legend has it that on their return Selkirk was introduced by Dr. Dover to Daniel Defoe who seized upon Selkirk's story as an excellent plot for his book.

Following his expedition Dover could not readily settle down but spent three years travelling on the continent. He seemed to have left an impression in Europe, for in the Opera "Onenia," by Ballorius (Geneva, 1760), there is a most laudatory dedication, in which Thomas Dover is extolled as one of the most distinguished physicians of the time.

At the age of sixty-one Dover settled down to practise in London where he was admitted licentiate of the College of Physicians and allowed to practise for six miles about Westminister. In order to gain eminence he turned to the time-honored custom of writing a book, publishing in 1733 "The Ancient Physician's Legacy to His Country"—being what he has collected himself in forty-nine years of practise and being designed for the use of all private families. The book seems to have been well received by the lay people for it "made a great noise in London and was the subject of almost every coffee house," but since it contained little of value beyond the prescription for Dover's powder it was read only by the uninformed.

In the section on gout appears the formula for his famous powder. "Take opium I oz., Salt-petre and Tartar vitriolated each IV oz., Ipecacuana I oz. Put Salt-petre and Tartar into a red hot mortar stirring them with a spoon until they have done flaming, then powder them very fine; after that slice in your opium, grind them to a powder, and then mix the other powders with these. Dose from 40-70 grains in a glass of white wine Posset going to bed (Posset is the whey which results from curdling milk by the addition to it of port or sherry), covering up warm and drinking a quart or three pints of Posset Drink while sweating them. Some apothecaries advised their patients to make their wills and settle their affairs before venturing upon so large a dose. However, Dover states, "I can produce undeniable proofs where a patient of mine has taken no less than 100 grains and yet appeared abroad the next day."

With the "good Dr. Sydenham," Dover prescribed the cold treatment for fevered patients. He argued to "lay aside Blisters and all heating and poisonous Powders" while he would, 'Have cold bathing grow as universal as inoculation." As resident pupil with Dr. Sydenham he writes, "whilst I lived with Dr. Sydenham I had myself the small-pox and fell ill on the twelfth day. In the beginning I lost 20 ounces of blood. He gave me a vomit but I find by experience that Purging is better. I went about by his direction until I was blind and then took to my bed. I had no fire allowed in my room, my windows were constantly open, my bedclothes were ordered to be layed no higher than my waist. He made me take twelve bottles of small beer acidulated with spirit of vitriol every 24 hours."

Dover's chief legacy to his country

was quick silver. Used as a specific in almost every disease he would order 1 oz. or 1¼ oz. crude mercury daily believing it to "relieve the patient of all veruncular disease, to open all obstructions and make a pure balsam of the blood." In derision he was called by his patients—"the quick-silver doctor." Dover was successful in exciting an active pamphlet war which for 20 years held up to discussion the powers of crude mercury. Dr. Turner of the College of Physicians hints that Dover's mercury treatment hastened the death of the celebrated tragedian Barton Booth to whom he had given in 5 days almost 2 pounds of Hg.

First Dover assaulted the apothecarie and then the physicians. During the 17th century there was a prolonged contest between the physicians and apothecaries; the former accusing the apothecaries of usurping their provinces, the latter continuing and justifying their usurption. In condemning the policy of the apothecaries Dover states "'Tis the writing physician only who has title to a fee," while explaining he was "always unviolably attached to the interests and welfare of the patient entirely regardless of the Gentleman's Unwarrantable Gains," while he constantly refused to vary his prescriptions merely to benefit the apothecaries who dispensed the medicines. The Royal College of Physicians was denounced as a "clan of prejudiced Gentlemen," while the unwary public are warned lest they take every graduate for a physician.

The only account of Dr. Dover's death appears in the London Magazine, April, 1742—"Dr. Thomas Dover, famous for administering quick silver to his patients, in his 85th year. The last years of his life were spent at Stanway house with his friend Robert Tracy to whom he dedicated his book."

So long as members of the medical profession prescribed ipecac and opium in the relief of disease the name of Thomas Dover, M.D., will remain immortal. We cannot fail to be vividly impressed by the romance of this spirited little Englishman who was successively practitioner, philanthropist, buccaneer and author, and who help make possible the creation of Robinson Crusoe.

Treatment of Influenza.

By ROBERT DAWSON RUDOLF, C.B.E., M.D., C.M. Edin., F.R.C.P., London.
Professor of Therapeutics.

Influenza is undoubtedly due to infection but the nature of this is still uncertain and hence specific therapy is unsatisfactory.

But if we cannot promise the suffers any radical treatment we can help them greatly in their struggle against this infection, and in many cases skilled medical care may make the difference between recovery and death.

Influenza itself is usually a slight disease, although in exceptional instances the patient may be very ill or even overwhelmed by toxaemia. The chief danger of the infection lies in the fact that it lays the sufferer open to sequelae of which by far the most common are respiratory.

The disease comes in great epidemics, which spread rapidly over whole continents and exact a terrible mortality. The infection is not influenced by climate and in the great epidemic of 1918-19 was equally prevalent and fatal in the tropics and in the arctic regions. In this epidemic which so paralyzed the different armies engaged in the Great War, the first wave came in the summer and was attended by almost no mortaliy. This "three-day

fever," as it was often called, temporarily knocked out whole regiments but the men were soon able to return to duty and often looked on their illness as almost a joke. Complications were practically unheard of. In October the next wave occurred and although many of the cases were as mild as before now sequelae were common and deaths from bronchitis and pneumonia were common. The same thing happened in the spring of 1919, and after that the disease gradually died away although sporadic cases still occurred for years and indeed never quite ceased. Lately there has been a recrudescence, amounting to an epidemic in many places.

It has often been noted that the mortality in these epidemics is chiefly among the young and previously healthy adults, and the explanation is probably that the others have had slight and often symptomless attacks ("subinfections" as Adami termed them), which have rendered them more or less immune. An objection to this theory is that bad attacks do not seem to confer any lasting resistence to further ones.

In an essay on treatment, etiology and morbid anatomy would be out of place, but a word on diagnosis.

Diagnosis. During an epidemic the recognition of cases or influenza is usually easy and the mistake is rather made of putting too many rather than too few cases under this heading. Almost everything tends to get labelled as "influenza" at such a time.

But at the commencement of a wave and later when this is waning, cases may easily be missed. It is often difficult to distinguish between a common "cold" and true influenza. The outstanding features that suggest the latter are the following:—Sudden onset (hence the name la grippe); headache and often pains in the back and limbs; frequent cough, dry and very irritating at first and later with often profuse purulent sputum; more or less fever and profuse sweating; a moist furred tongue, anorexia, nausea and even vomiting. Sometimes a slight erythematous rash suggests scarlet fever.

Along with some of these symptoms there is usually an unusual degree of prostration, which often suggests influenza in what looked at first like a common "cold."

Laboratory findings frequently are of value in excluding typhoid fever, the paratyphoids, acute tuberculosis, malaria, and rare conditions like meningitis and encephaliits. A low white blood count is the rule in the absence of complications. The finding of Pfeiffer's so-called **bacillus influenzae** is of no diagnostic value as this organism occurs with equal frequency in other infections.

There are several Types of Influenza depending upon the predominence of special symptoms. The following are the chief ones described:—

(1) The Nervous Type. Here headache is severe; there may be paralyses, neuritis or signs of meningeal irritation which may suggest meningitis. Spinal puncture will help to clear up the case. No doubt encephalitis and influenza are sometimes confused, the one with the other.

(2) Respiratory Type. There is nearly always some respiratory irritation, but this may be excessive and be the chief complaint. In many instances secondary infections of the respiratory system occur, and are, indeed, the chief cause of the mortality in influenza. The most common of these are with **pneumococci, micrococcus catarrhalis,** and various strains of **streptococci.**

(3) Alimentary Type. Here the stomach or the bowel or both bear the brunt of the attack, and nausea and vomiting and more rarely diarrhoea occur.

(4) Febrile Type. There is nearly always some fever in influenza but occasionally this is the chief symptom and the temperature may rise very high and may suggest typhoid or acute tuberculosis. The sudden onset is against these. Usually the pulse does

not hasten in proportion to the temperature.

In rare instances we meet with cases in which there is no rise in temperature.

The Complications of influenza are legion. Besides the respiratory ones, which are by far the most frequent, almost any organ may be involved. Otitis media is very common, and cardiac weakness often occurs and may last for long. In the cardiac cases the rate of the heart is usually fast, but occasionally bradycardia occurs.

Pulmonary tuberculosis frequently seems to date from an attack of influenza, but, on the other hand, it was often noted in military practice that distinct cases of phthisis came through influenzal attacks without any lasting exacerbation of their condition.

Environment. Although influenza occurs in any climate there is much evidence that chills tend to bring on complications, especially respiratory ones, and hence it is important to keep the patient warm in bed as long as there is any fever. The story that "He had a cold in the head; took a chilly journey and now has pneumonia" is all too common.

Diet. In the early stages the diet should be light and nourishing, with frequent warm drinks. Beyond this there are no special indications except in the alimentary type. Here the stomach may resent all food at first and for a day or two it is best not to press this, and then later feel one's way very cautiously.

During convalescence the patient will require feeding up and if the appetite lags a bitter stomachic such as quassia or nux vomica before meals is often of assistance. Cod liver oil is a most valuable food during the recovery from respiratory complications and should be given as soon as the stomach will stand it. If the patient has been used to taking any alcholoic beverages these may be allowed in the form of a bitter ale or stout or as a light wine.

Specific Treatment.

A. **Prophylactic.** The essential nature of the infection being uncertain no specific preventative therapy is possible. Inoculations with the **bacterium** pneumosintes (the possible infective agent along with Pfeiffer's bacillus) have been tried without any convincing results. But although we cannot specifically protect against influenza itself it is probable that considerable resistence can be set up towards the complicating organisms which after all are the chief cause of the mortality. A vaccine is used here containing the likely infective organisms, namely **bacillus influenzae, micrococcus catarrhalis,** the different strains of **pneumococci,** and haemolytic and non-haemolytic **streptococci.***

During an epidemic it is difficult for people to avoid the infection, but in some instances as in isolated institutions this has been successfully done. But where this is impossible it is well for people to at least avoid massive infection and hence as far as possible to keep out of crowded places and away from the vicinity of acute cases. Such cases are usually infectious for about a week.

The wearing of masks by those in attendance upon influenza patients has often been tried but with very doubtful results.

The impregnation of the air of public buildings with some antiseptic gas, usually chlorine, has had its advocates, but the results are not striking. Personally I believe that the frequent inhalation of aromatic antiseptics, such as menthol and eucalyptol, when near a case, lessens the chances of infection. The fumes of tobacco are believed by many to act in the same way.

During an epidemic the public will lessen their chances of taking the disease if they keep their general health in as high a state as possible by sensible dieting, plenty of fresh air and

*But several inoculations are necessary at some days intervals and hence the time for such protective therapy is before the influenza occurs.

sunshine and the avoidance of fatigue and chills.

B. **Curative.** There is no specific curative treatment of influenza. The body must elaborate its own immune defences, the while we endeavour to control the symptoms and sustain the vital forces.

Symptomatic Treatment. When the patient is kept warm in bed and on a light nourishing diet the most has been done for him. But symptomatically a great deal of relief may be given which will not only make him more comfortable but will help him in his struggle towards immunity.

When a great many different drugs are recommended in the treatment of any condition the young practitioner can conclude that none of them have any very decided action, and this is the case in the disease under consideration.

Oil of cinnamon was much used in the last great epidemic but without any decided benefit. 1 to 5 minims every 4 hours was the usual dose.

Salicin has often been recommended in massive doses (20 grs. every hour for 12 hours, then 20 grs. every two hours for the next twelve hours and after that less frequently as required). It certainly makes the patient more comfortable and seems to lessen the chances of complications.

Ammoniated tincture of quinine is a favorite with many, in drachm doses every four hours, or a combination like the following may be used:—

R/

Quin. Suphat gr. I.
Phenacetin. gr. II.
Salicin. gr. III. Ft. Capsul.
Sig. One every four hours.

The chief complaints being usually fever, dry, irritating cough and pains in various parts of the body, Dover's powder is useful, either alone, or in a combination like the following:—

R/

Pulv. Ipecac. Co.
Phenacetin. aa gr. II.
Acid. Acetylsalicyl. gr. IV. Ft. Capsul.
Sig. One every four hours.

This usually lessens the fever and produces considerable sweating, and also eases the cough.

Under some such treatment as the above the patient is usually (in the absence of complications) much better in two to four days, but convalescence is apt to be slow and he should not be in a hurry to expose himself to the chances of chills or great fatigue, as such may lower his resisting power so much that secondary infections may occur.

Suitable tonics and a change of air with all that this means are useful at this stage. In the respiratory cases cod liver oil is of special value.

In cases where convalescence is protracted a careful search should always be made for any possible focus of infection, this being specially apt to be situated in the sinuses or the tonsils.

Solid and Cystic Tumors of the Ovary.

By D. N. HENDERSON, M.B.

Ovarian tumours comprise two per cent of the pathological conditions encountered by gynaecologists. They occur from infancy to old age but are most common between the years of twenty and sixty, with the peak occurring during the time of sexual activity, twenty to forty years. Malignant tumours have the same age incidence as malignancy elsewhere in the body, being encountered at any age but most commonly after thirty-five years. The solid teratoma however, an extremely malignant tumor, occurs as

a rule before puberty. Thirty-five per cent of all ovarian neoplasms are cystic and belong to the cystadenoma group.

The histogenesis of ovarian tumours is very poorly understood. There are well recognized epithelial elements occurring in the ovary but many epithelial tumours are made up of cells which do not resemble any of these recognized pre-existing epithelial elements. Teratomata which embrace all three embryonic tissue layers are common to the ovary and have many interesting and fanciful theories of origin. Epithelial tumours of the ovary may arise first, from the germinal layer or surface epithelium, second from grandulosal cells of the Graafian folllicle and third from vestigial remains or cell rests found in the hilum of the ovary and called Pfluger's tubules.

Largely due to the confusion of histogenesis the classification of ovarian tumours shows wide variation in different text books. They are usually based on a combination of histogenesis, gross and microscopic appearance and fluid content. The following is such a classification :—

Retention cysts:

a. Germinal Inclusion.
b. Graafian Follicle.
c. Corpus Luteum, cysts and haematomata.
d. Endometrial.

Neoplasms of the Ovary:

a. Benign cystic.
   Simple serous cyst.
   Serous cystadenoma.
   Papillary serous cystadenoma.
   Pseudomucinous cystadenoma.
   Cystic teratoma.
b. Malignant cystic.
   Malignant papillary serous cystadenoma.
c. Benign Solid.
   Fibroma.
   Myoma.
d. Malignant solid.
   Adenocarcinoma.
   Sarcoma (all types).
   The rare forms such as Krukenberg, teratoma, granulosal cell carcinoma, etc.

To complete this classification one should add parovarian and inflammatory tubo-ovarian cysts, but these will not be discussed. All ovarian tumors cannot be definitely placed in the above classification. Many papillary tumours have solid areas, and solid carcinoma may undergo cystic degeneration. Sarcoma and carcinoma may be found together and a cystic teratoma may be malignant. It is often difficult and sometimes impossible to decide whether a papillary tumour is benign or malignant. Only the common ovarian neoplasms will be discussed.

Retention cysts—Germinal inclusion cysts are small and unimportant clinically. They are formed by an infolding of the germinal epithelium of the ovary and never reach a large size. Peri-oophoritis is the commonly accepted etiology of thees cysts.

Graafin Follicle cysts are extremely common and are encountered so frequently that they are almost looked upon as physiological. They rarely reach a size larger than 3 c.m., in diameter, occasionally one is encountered measuring as much as 7 c.m. They are usually multiple and bilateral, have a thin translucent wall and are filled with a clear watery fluid. They are lined by granulosal cells of varying thickness, depending on the size of the cyst and the resulting fluid pressure. Peri-oophoritis causing a thickening of the tunica albuginea is supposed to prevent rupture of the mature follicle. The ovum dies and a cyst results from the continued secretion of the granulosal cells. It seems well established also that ovaries which have their circulation disturbed, due to prolapse or some surgical procedure are very liable to become "cystic."

Many symptoms have been ascribed to follicular cysts and at one time oophorectomy was the treatment employed. At present it is believed that they are of little clinical importance. A large prolapsed cystic ovary may be

tender and give rise to dyspareunia or neuralgic pelvic pain. It is doubtful if they play any role in abnormal uterine bleeding or dysmenorrhoea.

Surgery is rarely required in treatment. If they are encountered at operation resection or needling is the procedure of choice.

Corpus Lutein cysts need only be mentioned. They are considered pathological when they measure over 3 c.m. in diameter. The cyst is filled with a clear yellowish fluid and lined by the yellow pigmented lutein cells. A haematoma differs only in its fluid content. Corpus lutein cysts and haematomata are usually associated with pelvic infections. The bleeding so commonly seen in subacute and acute salpingitis is attributed to infection of the corpus luteum with a resulting perversion of function. Multiple lutein cysts are encountered in hydatidiform mole and chorio-carcinoma. A corpus lutein haematoma which continues to bleed may simulate a ruptured ectopic pregnancy and requires the same treatment.

Endometrial or Samson's Chocolate cyst is a cyst lined by endometrical tissue which under the influence of the ovary menstruates monthly. The resulting blood forms the distending fluid of the cyst. It may be associated with endometrial implants elsewhere in the pelvis and presents symptoms associated with endometriosis.

Neoplasms of the Ovary. Simple serous cysts of the ovary are small, unilocular, pearly grey tumors, which occasionally reach the size of a man's head. They are lined by a columnar, cuboidal or flat epithelium depending on the fluid pressure. The wall is thin, transparent and made up of fibrous tissue in which ovarian stroma as a rule can be seen. The fluid content is watery and straw coloured.

The three types of cystadenoma have many common gross and microscopic characterizations. They are all multilocular and may reach a huge size. The walls show variation in thickness and colour, and have a bossed appearance. Large veins frequently run over the cyst wall and haemorrhage into the cyst content is common. As their names imply, they combine adenomatous tissue in the wall, and it is due to this fact that these cysts are multilocular. Not infrequently a cyst will present in one area the serous type and in another the papillary and in still another the pseudomucinous.

The Serous Cystodenoma is multilocular and usually unilateral. The wall is made up of fibrous tissue, ovarian stroma, glands and cystic spaces. These are lined by columnar or cuboidal epithelium with a round, deeply staining nucleus. The larger cystic spaces are lined by a similar epithelium, which is often flat, due to pressure and is not infrequently absent in many areas. The fluid content is watery, often colorless, but may be yellowish or reddish, due to blood pigment.

Papillary cystadenoma is a similar cyst except that the epithelium shows papillary projections into the lumen of the cyst, common type, or out through the cyst wall, the rare type. They usually are bilateral. The epithelium is of the type just described. The cells are arranged in a single layer and there is no breaking through the basement membrane. The papillary outgrowths may appear on the surface of the cyst and even on adjacent organs. Yet when the tumor is removed they disappear. This is rare, however. The benign papillary cystadenoma is in many cases extremely difficult to diagnose from the malignant variety.

The malignant form differs only in the excess of the papillary growths. Many solid areas of epithelial cells are seen with an epithelial lining, which shows variation in size and frequent nuclear figures. The fluid content of the cyst is usually blood stained. The malignant cyst is frequently densely adherent to bowel, omentum and anterior abdominal wall.

The Pseudomucinous Cystadenoma

can be differentiated from the serous type by the fluid content. The fluid is thick and tenacious, resembling raw egg white. It contains pseudomucin, a substance resembling mucin, but which is not precipitated by boiling with acetic acid. These cysts are usually unilaterally multilocular and attain a large size; cases having been reported where the cyst weighed as much as or more than the patient. They show the same adenomatous tissue in the wall. The glands and cystic spaces, however, are lined by a peculiar type of epithelium not unlike that of the cervix. The cells are high columnar, with a clear, pale pink cytoplasm and a small nucleus situated near the base of the cell. The cells are ciliated and only rarely are papillary projection seen. These cysts rarely become malignant. However, they are associated with an interestng condition known as "pseudomyxomta peritoneo." This is due to the spilling of the cyst contents into the abdominal cavity along with a few of the pseudomucinous secreting cells. Here they grow, secrete pseudomucin and set up a sterile peritonitis. The abdominal cavity soon becomes the site of fibrous adhesions and is filled with this jelly-like pseudomucin. Nothing can be done for these patients, and they die a slow, wasting death unless some intercurrent infection or intestinal obstruction brings a more rapid termination.

Malignant papillary cystadenoma has been described under the benign type.

Solid Adenocarcinoma of the ovary is usually a bilateral growth, first confined to the ovary, but rapidly spreading beyond its borders. Pseudogland formation and papillary surface growths are common. It is not unusual for a malignant papillary cyst to be found in one ovary and a solid carcinoma in the other. Extension is rapid and the tubes, ovaries and uterus are soon bound together in a fixed mass.

One of the rarer forms of solid carcinoma of interest is the Krukenberg tumor, a solid carcinoma of the ovary which is a secondary growth from a primary situated in the stomach. Frequently the secondary growth is first discovered and well established before the primary has been diagnosed. These tumors are bilateral.

Fibroma of the ovary is a solid tumor which rarely reaches a size larger than a man's head. It is a fibrous tissue growth with a white whorled surface on section. As with most solid tumors of the ovary it is usually associated with ascites.

Sarcoma of the ovary is rare and all types have been described.

Teratomata are usually interesting tumors. They are solid or cystic. The solid form is rare and occurs as a rule in young children and is extremely malignant. The cystic variety which are commonly encountered and called dermoids, are frequently bilateral. They are filled with a thick gelatinous fluid which when cooled becomes solid. They do not reach a large size. The so called embryoma, a thickened portion of the cyst wall situated near the base of the cyst, contains skin, hair, teeth, bone, etc. Elsewhere the cyst is lined by granulation tissue in which foreign body giant cells are seen. Malignant change may occur in any of the tissues in the embryoma.

Symptoms.—The symptoms present depend on the size and position of the ovarian neoplasm, whether it is solid or cystic, malignant or benign.

With a benign cystic tumor the commonest complaint is one of an enlarging abdomen. Pain is frequent, being due to pulling on adhesions with change of position, or partial rotation of the pedicle. A cyst situated in the pelvis will frequently cause more symptoms than a large one which has grown into the abdomen. Pressure on the bladder causes frequency of micturition and on the rectum constipation. When the cyst becomes incarcerated all pressure symptoms are increased and pain is considerable. There is as a rule little disturbance of menstruation. Amenorrhoea followed by menorrhagia is very

suggestive of malignant change. A large cyst which has grown out of the pelvis commonly causes no symptoms until some secondary change occurs. Very large cysts cause embarrasment of respiration, marked pressure symptoms on the stomach, bowel, bladder and vessels. Oedema of the legs and vulva with prominence of the abdominal veins is common. Prolapse of the uterus due to pressure is not rare. Large cysts soon cause general debility and cachexia.

Dermoid cysts have a greater tendency to cause amenorrhoea and are more liable to infection and torsion of the pedicle. They have a tendency to lie in front of the uterus in the utero-vesicle pouch and thus cause rather marked frequency of micturition.

Malignant cystic tumors may present any of the above symptoms, but pain is more marked, due to the frequent presence of adhesions. Ascites is the rule and general debility and cachexia are rapid. The small malignant papillary and solid carcinoma frequently produce extreme ascites. Pelvic pain is the rule and menorrhagia is common.

Solid fibroma is frequently bilateral and associated with amenorrhoea. These tumors are heavy, causing a dragging type of pain and discomfort. Due to their weight they frequently undergo torsion of the pedicle. Ascites is common.

The Diagnosis of Ovarian Neoplasms as a rule presents no great difficulty. In the small variety when the tumor is still pelvic, nothing can be felt on abdominal examination. On pelvic examination the first essential is to outline the body of the uterus. A normal ovary on one side, absent on the other and replaced by a round cystic structure, makes the diagnosis easy. If the cyst is frequently moveable it can be well outlined and any irregularity noted, while any particularly firm areas may suggest dermoid or carcinoma. Frequently the uterus is pushed to one side or the other, or back into the hollow of the sacrum when the tumor lies in front. A solid tumor, as a rule, when small maintains more or less the shape of the ovary, has not the round, smooth outline of a cyst, is frequently bilateral and is as a rule accompanied by ascites.

With the large cystic tumors the abdomen is rounded with the lower portion more prominent. A wave of fluctuation is usually evident and the tumor is dull to percussion with tympany in the flanks if ascites is not present. On pelvic examination the uterus may be prolapsed due to pressure and the fluctuant mass can be felt pushing down the pelvic organs. In a moderate-sized tumor the pedicle can be felt, indicating from which side the cyst arises.

Malignancy is suspected when the growth of the cyst has been rapid, when ascites is present and cachexia evident. In the solid carcinoma the mass is fixed and hard. Frequently the tumor is bilateral and the fornices hard and irregular, while the uterus is fixed between the two lateral masses. These findings may be mistaken for a marked pelvic inflammation and cellulitis.

Often examination is difficult due to marked ascites. Paracentesis abdominis may be done before examination. In any case of doubt on pelvic examination the administration of a general anaesthetic of gas and oxygen will give the required relaxation for proper and careful examination.

Accidents indidental to ovarian tumors.—Rotation of the pedicle with strangulation and hemorrhage into the cyst wall and contents is the commonest accident incidental to ovarian cysts. Heavy tumors such as dermoids and fibroma, also cysts free from adhesions and with a long pedicle, are particularly liable to this accident. The patient suffers sudden pain, usually vomits and shows signs of shock. If the strangulation is complete gangrene and infection occur and the patient dies from peritonitis. On examination the tumor is palpated, found to be very tense, and as a rule extremely tender.

Fever is usually present. Treatment is surgical.

Infection of ovarian cysts is now relatively rare since the practice of tapping has largely been discontinued. Infection usually occurs following full term labour, abortion, or tubal inflammation. The symptoms are those of pelvic inflammation and the condition requires palliative or surgical treatment, depending on the individual case.

Rupture of ovarian cysts is a rare complication, being due to trauma, sudden increase of the intracystic pressure or a thinning of the cyst wall with spontaneous rupture. Symptoms of rupture vary depending on the fluid content and size of the cyst. Hemorrhage may occur, producing marked shock and requiring immediate operation. Rupture of a malignant cyst leads to complete abdominal dissemination of the growth. Fluid from a serous cyst is readily absorbed, while that from a pseudomucinous cyst sets up a sterile peritonitis, as already described.

The treatment of ovarian neoplasms is surgical. Our inability to determine what type of cyst we are dealing with, the possibility of axial rotation infection or malignant change in a benign tumor makes operation the procedure to be recommended.

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